WHITE RUSTS

A REVIEW OF ECONOMICALLY IMPORTANT SPECIES

G.S. SAHARAN

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White Rusts

A Review of Economically Important Species

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1. INTRODUCTION

White rusts or stagheads are caused by several species of the fungus Albugo, which is the only genus in the Family Albuginaceae in the Order Peronosporales of Class Oomycetes. These diseases, being serious and widespread on a large number of host plants,have long been popular areas of study for mycologists and plant pathologists (1, 5, 7, 21, 23, 28, 37, 40, 51, 54, 57, 59, 60, 77, 79, 87, 88, 90, 92, 99, 100, 105, 107, 112, 113, 118, 120, 144, 152, 155, 159, 161, 163, 165, 175, 176, 179, 180, 187, 189, 197, 203, 208, 215, 221-223, 227, 231, 236, 242, 243, 247, 248, 251-256, 262-266, 268, 273, 275, 278, 280).

Among the white rust diseases affecting several crops, the one caused by *Albugo candida* (Pers. ex. Hook.) O. Kuntze on Cruciferae and some species of the Capparidaceae and Cleomaceae is the most widespread. Other economically important diseases caused by related species of Albugo include 1) white rust of spinach, *A.. occidentalis* Wilson (179, 180, 273, 278); 2) white rust of sweet potato, *A. ipomoeae-panduratae* (Schw.) Swingle (28, 46, 79); white rust of water spinach, *A. ipomoeae-aquaticae* Sawada (88, 185); 4) white rust of salsify, *A. tragopogonis* (DC) Gray (278); and 5) white rust of sunflower, *A. tragopogonis* (DC)S.F. Gray (149, 155, 195). Of lesser importance are white rust of Amaranths, *A. bliti* (Biv.-Bern.) Kuntze (147); white rust of Portulaca, *A. quadrata* (Waller) Comb; and white rust of Mesembryanthemum, *A. mesembryanthemi* sp. (5).

2. THE DISEASE

a) History: Much of the initial research on white rust diseases focussed on mycological aspects of the pathogen rather than on the disease itself. The first species of white rust discovered on cruciferous plants was named *Aecidium candidum* by Persoon in 1791. Later Persoon in 1795, proposed the genus *Uredo* to include this species and others. In his "Synopsis Methodica Fungorum" (165) the genus *Uredo* is divided into four sections, the third of which is *Albugo*, characterized by having white spores, and including *Uredo candida* (with forms *U. thlaspeos*, *U. tragopogi*, *U. alysii*

and *U. cheiranthi*). Recognizing its affinities with the Phycomycetes, Gray (71) defined *Albugo* as a separate genus containing three species, *Albugo cruciferarum*, *A. tragopogi* and *A. petroselini*. The group was not mentioned by Fries (64) in "Systema mycologicum". Leveille (133) also did not recognize *Albugo* and erected a new genus *Cystopus* in 1847 to include *Uredo candida* Pers., *U. cubica* Mart, *U. portulacae* DC, *U. bliti* Bivon, and *U. floriformis* Merat, etc. Oospores and sex organs were not described until deBary (41) who revised the genus to include the perfect stage in 1865. Kuntze correctly revised the name *Albugo* in "Revisio Genera Plantarum" in 1891. Ramsbottom (181) and Wakefield (265), when reviewing the genus causing white rusts, recognized *Cystopus* deBary because deBary was the first to describe the perfect stage. Baker (5), while describing the genus *Albugo* in New Zealand, upheld the validity of *Albugo* against *Cystopus*. Pathological development of white rust diseases has been reviewed by Butler (21), Butler and Jones (23), Chupp (27), Eberhardt (51), Heald (81), Kolte (113), Mukerji (149, 150), Saharan (187), and Walker (266). The chronological development on each aspect of white rust diseases affecting different host plants is reviewed in the following pages.

b. Host species affected: *A. candida* infects a large number of host plants in Aizoaceae, Cruciferae, Capparidaceae and Cleomaceae, including leaves of *Reseda alba* (98, 150, 266, 267). According to Biga (13) *A. candida* infects plants of 241 species in 63 genera of Cruciferae. Hosts amongst the cultivated crops include *Brassica alba, B. oleracea* var. *capitata* (cabbage), *B. oleracea* var. *botrytis* (cauliflower), *B. rapa* (turnip), *B. juncea* (Indian mustard), *B. nigra* (Black mustard), *B. rugosa, B. chinensis, B. pekinensis, B. campestris* (turnip rape), *B. campestris* var. Toria, *B. campestris* var. Brown Sarson, *B. campestris* var. Yellow Sarson, *B. napus, B. tournefortii, Raphanus sativus, Armoracia lapathifolia* (horseradish), *Lepidium sativum* (cress), *Rorippa nasturtium* (watercress), *Cheiranthus cheiri* (wallflower), *Matthiola incana* (stocks), *Raphanus raphanistrum,* and many weeds (21, 81, 143, 148, 169, 170, 266, 272, 274). *Capsella bursa-pastoris* (Shepherd's purse), *Lepidium*

virginicum (pepper grass) and *Sisymbrium officinale* are the most common weed hosts (68, 81).

Albugo ipomoeae-panduratae infects members of the Convolvulaceae, especially *Ipomoea* and *Convolvulus* spp. Economically important hosts include I*pomoea batatas* (sweet potato), *I. pes-tigridis* and probably *I. aquatic* (= *I. reptans*, water spinach). *i. horsfalliae, I. purpurea*, and *Calonyction aculeatum* are amongst ornamentals attacked by the fungus (151). Legumes are infected by *Albugo mauginii* (104), while members of the Compositae are infected by *Albugo tragopogonis* (98).

Ten species of *Albugo* infecting several cultivated and wild hosts have been reported from India (249). In addition, Wilson (278) listed several wild hosts from 12 different families including Cruciferae, Portulacaceae, Chenopodiaceae, Amaranthaceae, Convolvulaceae, Boraginaceae, Piperaceae, Caryophyllaceae, Ambrosiaceae, Cichoriaceae, Carduaceae and Allioniaceae.

c. Geographical distribution: White rust on cultivated *Brassica* and other cruciferous species is prevalent in several localities throughout the world. Although the list is not exhaustive, countries where the disease occurs include the U.K. (7), U.S.A. (266), Brazil (261), Canada (31, 72, 167), Germany (110); India (26), Japan (85), Pakistan (166), Palestine (182), Romania (195), Turkey (19), Fiji (161), New Zealand (76) and China (281).

White rust of sunflower occurs in Russia (155), Uruguay (183), Argentina (194), Australia (145, 229), and in many other countries (100). White rust of salsify occurs in Australia, Canada, U.S.A., S. America, Europe, Asia and Africa (278). White rust of water spinach is a serious disease in India and Hong Kong (88, 185) and affects spinach in Texas (273, 275).

White rust of *Ipomoeae* and *Convolvulus* spp. is widespread in tropical and sub-tropical regions, including North America (USA), South America (most countries), Africa (Morocco, Sudan and countries south of the Sahara), S.W. Europe (S. France, Italy, Malta), S.W. USSR (Azerbaijan, Turkmenistan, Uzbekistan), Asia (India, Pakistan and

Far East), Australia (Queensland), Fiji, Guam, Jamaica, Puerto Rico, West Indies, Haiti, Argentina, Brazil, Trinidad, and E. China (79, 151).

d. Symptoms: Symptoms resulting from the local or leaf phase and the systemic or staghead phase of infection in different crucifers have been described in detail by Butler (21), Heald (81), Butler and Jones (23), Kolte (113), Vasudeva (248) and Walker (266). White pustules (sori) of variable size and shape are formed on leaves (Figure 1), stems, inflorescences and pods. Sori are initially discrete, but later coalesce to cover whole plant organs. Distortion and hypertrophy of the affected host tissue is frequent (Figures 2,3). The stem and inflorescence axis may become thickened to many times their normal diameters. Leaves may also become thickened, with edges rolled to the lamina. The floral parts persist and show hypertrophy and/or hyperplasia. Maheshwari *et al.* (141) described structures and development of galls induced by *Albugo* in the inflorescence axis of *Brassica juncea*. On horseradish the first visible symptoms are the appearance of light yellow areas on the upper surface of leaves followed by creamy white blisters forming on the lower surface in the form of concentric rings (57, 99). Gall formation on roots of radish were described by Fisher (59).

Initial infection of water spinach (*Ipomoeae reptans*) appears as chlorotic flecks. White sori (5-6 mm-diameter) may eventually cover the whole leaf. Necrotic areas are formed after the asexual spores (sporangia) are shed. Cerebriform, stem galls occur on the stems (2-3 times the stem diameter), and plants that have root galls may be stunted. Bud galls which develop after systemic infection via axillary buds, grow into thick shoots with compressed internodes. Numerous systemically infected lateral branches and leaves that are distorted and reduced in size are also formed. Sori form on the surface of bud galls but not on cerebriform galls. Flower buds are swollen and aborted (88, 185, 274).

On sweet potato (*Ipomoeae batatas*), in addition to white pustules on leaves and stems, distortion and galls may also occur and galls may be formed on tubers (79).

Figure 1. White sporangial pustules (sori) on the undersurface of leaves. **Figure 2.** Hypertrophied inflorescences (stagheads) on *Brassica campestris* cv. Torch produced in field plots following infestation of seed with oospores. **Figure 3.** Stagheads on *B. campestris* cv. Torch resulting from flower bud inoculation in growth room. (Can. J. Plant Sci. 60: 267-271, 1980).

Singh (215) described the anatomy of galls induced by *Albugo ipomoeae-panduratae* on *Ipomoeae pentaphylla*.

e. Damage or Yield Loss: In several cultivated crops, earlier researchers (27, 110, 138) have reported only minor yield losses from white rust. However, with the passage of time white rust has become a major problem in cruciferous crops in many countries; the staghead phase causes heavy yield losses depending on the intensity of infection. Yield losses in rapeseed in north central Alberta were 1-2% in 1971 (8), while in Manitoba losses of 30-60% were reported in severe white rust infected fields (11). Petrie and Vanterpool (171) reported yield losses of up to 60% due to *A. candida* staghead infection in rapeseed in Saskatchewan. Harper and Pittman (78) determined the relationship between the severity of systemic infection of the stem and the seed yield of rapeseed using an equation for assessing yield losses under field conditions (% yield loss = 0.952 X % stems infected systemically). In Canada, Petrie (167) reported loss estimates from hypertrophied inflorescences in *B. campestris* of 1.68, 4.13 and 2.43 million dollars respectively for 1970-72. In western Australia, Barbetti (6) estimated annual yield losses of 5 - 10% due to stagheads in rapeseed.

Mixed infections of *Albugo* and *Peronospora* in *B. juncea* cause yield losses of 17-32% in India (3). Losses in yield due to mixed infections of downy mildew and white rust in *B. campestris* var. Toria is about 34% if the average length of the individual hypertrophied racemes is 10 cm (117). To estimate the losses due to mixed infections of white rust and downy mildew, or white rust alone, Kolte *et al.* (117) suggested the following formula:

$$Q = A - \frac{(B \times C)}{A} \times 100$$

where Q = % yield loss, A = average actual or expected yield of a healthy plant, B = average expected yield from the diseased raceme, which is equal to actual average yield from the corresponding length of a healthy raceme, and C = number of diseased racemes per plant. Saharan, *et al.* (190) estimated yield losses of 23 - 54.5% in late

sown Indian mustard cv. RH-30. Recently, Saharan and Lakra (192) and Lakra and Saharan (129) estimated yield losses in *B. juncea* cv. Prakash due to leaf and/or staghead infections of *A. candida*. In their study losses were correlated with intensity of infections in the crop with leaf and staghead phases combined and in isolation. This study has clearly shown the effect of each category of infection on different parameters contributing to yield; e.g., number of branches/plant, number of pods/branch, pod length, number of seeds/pod, 1000 grain weight and total yield per plant. The plants having the highest leaf phase and staghead phase infections suffered yield losses of 27.4 and 62.7% respectively, but the combined infection of both these phases reduced the yield by 89.8%. To estimate the losses on yield due to each phase of white rust infection a prediction equation has been suggested: $Y = a_1x_1 + a_2x_2$, where Y = % loss in yield, $a_1 = 0.437 \times _1$ and $a_2 = 1.176 \times _2$ are constants, and x_1 and x_2 are disease indices on leaf and staghead phases respectively.

f. Association: *Albugo candida* is frequently associated with the downy mildew pathogen, *Peronospora parasitica*. Numerous instances are known of considerable damage from combined infection (23, 139, 193, 197, 246). In India, mixed infections of downy mildew and white rust are common on *B. juncea* (4, 187). *A. candida* appears first on leaves (4), which predisposes the host tissues to infection by *P. parasitica*. The intensity of mixed infections varies from 0.5 - 35%, depending on local weather conditions (3, 187). The hypertrophied and malformed inflorescences of *B. juncea* infected with *A. candida* are usually covered heavily with the white powdery growth of the downy mildew fungal spores (25, 187). *Albugo candida* alone, on artificial inoculation of flower buds, induces typical hypertrophy of the inflorescence (Figure 3) (255). However, whether *Peronospora parasitica* alone induces hypertrophy of the inflorescence in *B. juncea* or other hosts remains to be confirmed. The role(s) played by *A. candida* and *P. parasitica* in induction of mixed infections of inflorescences in the field has yet to be determined.

Over twenty species of fungi, including several pathogens of crucifers, have been

found in association with hypertrophies of the inflorescence (stagheads) and stem and pod blisters produced on turnip rape (*B. campestris*), wild mustard (*B. kaber*) and false flax (*Camelina microcarpa*) by *A. candida*. The most prevalent fungal associates of *Albugo* are *Peronospora parasitica, Alternaria alternata, Fusarium roseum, F. accuminatum, F. equiseti, Alternaria raphani, A. brassicae*, and *Cladosporium* sp. (169).

3. THE PATHOGEN

a. Taxonomy and Morphology

Albugo (Pers.) Gray, 1821.

= *Uredo albugo* Persoon, 1801

= *Erysibe sensu* Walker., 1833

= *Cystopus* Leveille, 1847

= *Cystopus* deBary, 1863

Biga (13) re-examined the morphology of the sporangia and constructed a key, in Italian, to differentiate species of genus *Albugo*. Wilson (278) described the North American species, Savulescu (195) the Romanian species, and Wakefield (265) gave a historical note on the genus in her account of the species in South Africa. Morphological details of *Albugo* on crucifers were given by Togashi *et al.* (241). Baker (5) upheld the validity of *Albugo* against *Cystopus*. Waterhouse (267) described the species of *Albugo* on the Aizoaceae in the U.K. An account of Indian species was given by Butler and Bisby (22) and Safeeulla (184). Burdyukova (20) described the Russian species of *Albugo*. Species that have attracted attention as pathogens are as follows:

i) *Albugo candida* (Pers. ex. Hook.) O. Kuntze.

= *Aecidium candidum* Pers.

= *Uredo candida thlaspeas* Pers.

= *Uredo candida* (Pers.) Fr.

= *Cystopus candidus* (Pers.) Lev.

Mycelium intercellular with small globose to knob-like haustoria, one to several in each host cell. Sori white to rarely pale yellow, prominent, deep seated, variable in size and shape, often confluent. Sporangiophores hyaline, clavate, thick-walled, especially towards the base, 30-45 x 15-18 µM diameter. Sporangia arranged in a basipetal chain, globose to oval, hyaline with uniform thin wall, 12-18 µM diameter. Oospores globose, chocolate-brown, 30-55 µm (generally 45 µm with oogonial wall up to 60 µM), epispore thick, verrucose to tuberculate, or with low blunt ridges which are often confluent and irregularly branched, and sometimes seemingly smooth (90, 149, 150, 278). Biga (13) divided this species into two varieties on the basis of sporangial size.

ii) *Albugo ipomoeae-panduratae* (Schwein.) Swing.

= *Aecidium ipomoeae-panduratae* Schwein.

= *Aecidium ipomoeae* Schwein.

= *Cystopus ipomoeae-panduratae* (Schwein.) Stev. and Swing.

Mycelium intercellular with typical knob-like haustoria. Sori amphigenous or caulicolous, white or light yellow, prominent, superficial, 0.5-2.00 mm round, arranged concentrically, often confluent. Sporangiophores hyaline, club-shaped, unequally curled at base, 30 - 40 µm x 12-15 µM. Sporangia hyaline, rectangular, short cylindrical, the terminal more rounded, 12-20 µm x 12-18 µM. Wall with distinct equatorial thickening. Oospores caulicolous, spherical, light yellowish-brown, 25-40 µm (sometimes up to 45 µM, 55 µM with oogonial wall), epispore papillate or with irregular, more or less curved ridges (90, 151, 278). Biga (13) placed two varieties under this species on the basis of sporangial size.

iii) *Albugo tragopogonis* (DC). S.F. Gray.

= *Uredo candida* ß *tragopogi* Pers.

= *Uredo tragopogi* (Pers.) DC.

Mycelium intercellular, hyaline, with globose vesicular haustoria with small stalks. Sori (pustules) hypophyllous or caulicolous, prominent, deep seated, white or yellowish, pulverulent, rounded, or elongate, 1-5 x 1-8 mm. Sporangiophores hyaline, club-

shaped, 40-55 µM x 12-16 µM. Sporangia hyaline to light yellow, short-cylindrical to spherical-cuboid, the terminal larger, oval and less angular than the lower ones, wall with an equatorial thickening, 18-24 µM x 12-20 µM. Oospores spherical, dark brown to almost black at maturity, 44-57 µM (sometimes up to 68 µM with oogonial wall), epispore reticulate, densely covered by low, tuberculate or spinulose warts, meshes (areolae) 2-3 µm across (149). Savulescu (195) divided this species into two varieties on the basis of the size and shape of sporangia. Biga (13) made five varieties on the basis of host and size of sporangia. It is distinguished from other species of *Albugo* in having cylindrical sporangia and oospores with reticulate epispore tuberculate at their angles.

iv) *Albugo ipomoeae-aquaticae* Sawada.

Sori hypophyllous and on stem or inflorescence, round to irregular, 1-3-mm diameter, up to 7 mm long, dull white, becoming pulverulent, host sometimes distorted. Sporangiophores hyaline, clavate, 32-72 µM long, 18-23 µM wide. Sporangia catenulate, uniform size, globose to cuboid, hyaline 16-23 x18-26 (av. 19 x 21) µm, enclosed by a smooth, uniformly thickened membrane. Oogonium globose, subglobose or obvate with irregular patterns on the inner surface, 52-80 µM-diameter when globose or 64-89 µM x 48-68 µM when otherwise shaped, containing one oospore. Oospore globose, hyaline, smooth, 39-48 µM-diameter, the wall originally thin but becoming 6-8 µM thick after conjugation (53, 90, 96, 185, 198, 199).

A taxonomical and morphological account of 13 North American species of *Albugo* infecting different hosts, along with a key to the species, has been given by Wilson (278).

b. Structures and Reproduction

The members of the Albuginaceae are distinguished from those of related families by the formation of the asexual sporangia in basipetal chains.

1. Mycelium and haustoria

The non-septate and intercellular mycelium of *Albugo* species feeds by means of

globose or knob-shaped intracellular haustoria. The details of haustorial formation and development has been given by Berlin and Bowen (10), Coffey (29, 30), Davison (38), Fraymouth (62) and Wager (263).

2. Sporanglophore and sporangia

The mycelium soon organizes the characteristic groups of sporangiophores which develop beneath the epidermis, raising it to make whitish pustules or extended blister-like areas due to the merging of adjacent sori. As soon as the covering epidermis ruptures, the sporangia are set free (81, 263, 266). When a pustule or sorus forms, numerous short sporangiophores arise from the mycelium in a closely compacted palisade layer beneath the epidermis and at a right angle with it. The first sporangium is formed at the tip of the sporangiophore, and others are formed successively beneath. Pads of gelatinous material formed between successive sporangia function as disjunctors. The sporangiophores are short, basally branched, club-shaped and give rise to simple chains of sporangia. The number of sporangia produced are indefinite. They are formed in basipetal succession; that is, the sporangiophore forms a cross-wall or septum, cutting off that portion which is to become a sporangium. This gradually takes on the characteristic form: the sporangiophore increases in length, a second sporangium is cut off and the process continues, resulting in the simple chains of multinucleate sporangia. As sporangial production continues, the older, terminal portions of the chains break, releasing the individual sporangia. The sporangia are hyaline and nearly spherical. They germinate by the formation of zoospores and, on rare occasions, by means of a germtube (81, 263, 266, 280).

3. Zoospores

The production of zoospores was first recorded by Prevost in 1807 (177). Tulasne in 1854 (244) and Hoffmann in 1859 (89) were unable to confirm Prevost and described germination by germ tubes. Melhus (144) mentioned that DeBary (39) described for the first time the details of zoospore formation.

Sporangia absorb water and swell. Vacuoles in the granular protoplasm form and

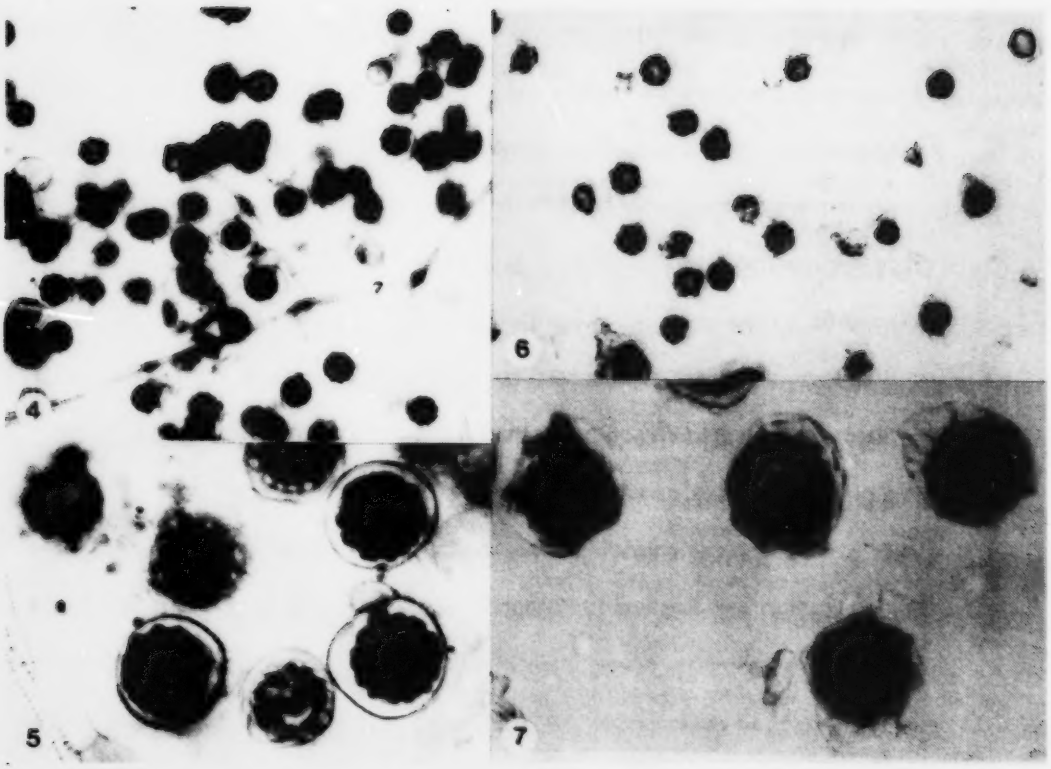
then disappear, and finally 4-12 polyhedral portions of the protoplasm are delineated by fine lines. In the meantime, an obtuse papilla is formed at one side of the sporangium, which now swells and opens. The zoospores, still immobile, emerge usually one by one, with final cleavage sometimes following complete emergence of the sporangium's contents. The flagella soon become apparent by an oscillatory motion of the entire zoospore mass. The slightly concave-convex zoospore contains a disc-like vacuole on one side, near which are attached two flagella, one short and one long, by which the zoospore soon detaches itself from the mass and swims away if liquid is present. Zoospores soon come to rest, absorb their flagella, encyst and germinate by the formation of a germ tube. If this germination occurs on a susceptible host, the germ tube will penetrate through a stoma to form an intercellular mycelium (81, 263, 266).

4. Sexual Organs

The oogonia and antheridia are formed from the mycelium in the intercellular spaces of the host, particularly in a systemically invaded tissue (264). Oogonia are globose, terminal or intercalary and each contains about 100 nuclei. The contents of an oogonium are clearly defined into a peripheral zone of periplasm and a single central oosphere. Antheridia are clavate, each containing 6 to 12 nuclei, and are applied to the sides of an oogonium (81, 82, 266).

5. Gametogenesis, Fertilization, and Oospore Formation

The classical information on the process of gametogenesis, fertilization and oospore formation in different species of *Albugo* has come from the exhaustive studies of Wager (264), Davis (37), Stevens (221-224) and Thirumalachar *et al.* (237). One or more antheridia come to occupy a position close to an oogonium. There are two types of egg organization within an oogonium. In certain species (*A. candida*), the protoplast becomes differentiated into a peripheral or external zone, the periplasm, which contains many nuclei, and a central mass, the egg cell or ooplasm, which contains a single nucleus. In other species (*A. bliti, A. portulacae, A. tragopogonis*), the central ooplasm remains multinucleate. The antheridium, which is a multinucleate cell, produces a



Figures 4-7. Oospores of *Albugo candida* race 7 in detached infected leaves of *Brassica campestris* cv. Torch **(Figures 4 and 5)**; and in naturally infected stagheads of *B. campestris* cv. Torch **(Figures 6 and 7)**.

short, tube-like outgrowth, the fertilization tube, which penetrates the periplasm and comes in contact with the egg. The antheridial or male nuclei are discharged through this tube into the egg cell. In the uninucleate egg, the female nucleus fuses with a single male nucleus, and in the multinucleate egg, female and male nuclei fuse in pairs. This nuclear union constitutes the process of fertilization (81, 266).

Following fertilization, the egg is gradually transformed into a thick-walled oospore (Figures 4-7, 9). The periplasm is absorbed, the oospore wall darkens and thickens and develops characteristic external ridges, reticulations or knobs, while the interior of the oospore becomes filled with an abundance of reserve food in the form of oily or fatty globules. The fully developed oospore lies within the old empty oogonial cell. The oospores are released only by the weathering and decay of the host tissue in which they were formed (81).

6. Oospores

The characteristics of oospores are a useful criteria for distinguishing species of *Albugo*. According to Wilson (278), the species fall into two well-defined groups. The first group includes *A. candida, A. ipomoeae-panduratae, A. lepigoni, A. sibirica* and *A. tropica.*, in which the epispore of the oospore is tuberculate or ridged (Figures 4-7). Zalewski (280) and Stevens (223) confirmed that this is the more specialized group, where there is complete development of the epispore with cytological phenomena. The second group is characterized by a reticulate epispore and contains the remaining species in which oospores are known. In *A. bliti* and *A. platensis* the reticulations are very evident, and the areolae are deep and unoccupied by elevations. The pattern is often imperfectly developed. In *A. tragopogonis* and *A. swertiae* the areolae are not as deep and the reticulations are crested at their angles with more or less prominent tubercles. In *A. portulacae* tubercles form within the areolae while the reticulations themselves are similar to those of *A. bliti.* Unique within the genus is *A. occidentalis*, in which the epispore is finely reticulate and the areolae so shallow as to give the impression of pits rather than reticulations (278). According to Zalewski (280) the

reticulate oospores have a less perfectly developed epispore which reaches its highest development in *A. tragopogonis*. Stevens (223) confirmed this arrangement of species.

c. Life Cycle

The symptoms of general or systemic infection of *A. candida* are distortion, hypertrophy, hyperplasia and sterility of inflorescences (stagheads) of the host plant (Figures 2,3,8A). When ripe, stagheads are almost entirely composed of brown, thick-walled oospores (Figures 4-7,8B) which are known to survive dry storage for a period of over 20 years (251). Oospores, carried on seed, or overwintered in soil, are an important source of initial infection in the field. After a resting stage, oospores germinate by the production of one or two simple or branched germ tubes (Figures 10-15), or by release of 40-60 zoospores from either sessile or terminal vesicles (251) (Figures 8C, 16,17,18). When the vesicle wall bursts, the zoospores are liberated, they swarm, encyst, and finally germinate by germ tubes (Figures 8D,E). Germ tubes from zoospores penetrate the host cotyledons/leaves through stomata (Figure 8F). The mycelium is intercellular and feeds by means of small, capitate, globose or knob-shaped haustoria which penetrate the host cell walls through minute perforations. Hyphae grow around palisade mesophyll cells as a downward spiral penetrating the individual cells with as many as 14 haustoria per cell (252). The coenocytic mycelium branches profusely and eventually fills all the available intercellular spaces. At a certain stage of maturity, short, club-shaped sporangiophores are developed from a dense layer of mycelium which shows a characteristic branching pattern not common in earlier stages of vegetative mycelial growth (252). The sporangiophores are borne in close proximity to one another in solid layers immediately below the epidermis of the host (Figure 8G). Each sporangiophore gives rise to several sporangia in succession with the oldest at the tip of the chain and the youngest at the base (Figure 8G). Both the growth of the mycelium and the production of an enormous number of sporangiophores and sporangia exert a pressure from below on the host epidermis causing it to break. The released sporangia form a white crust on the surface of the

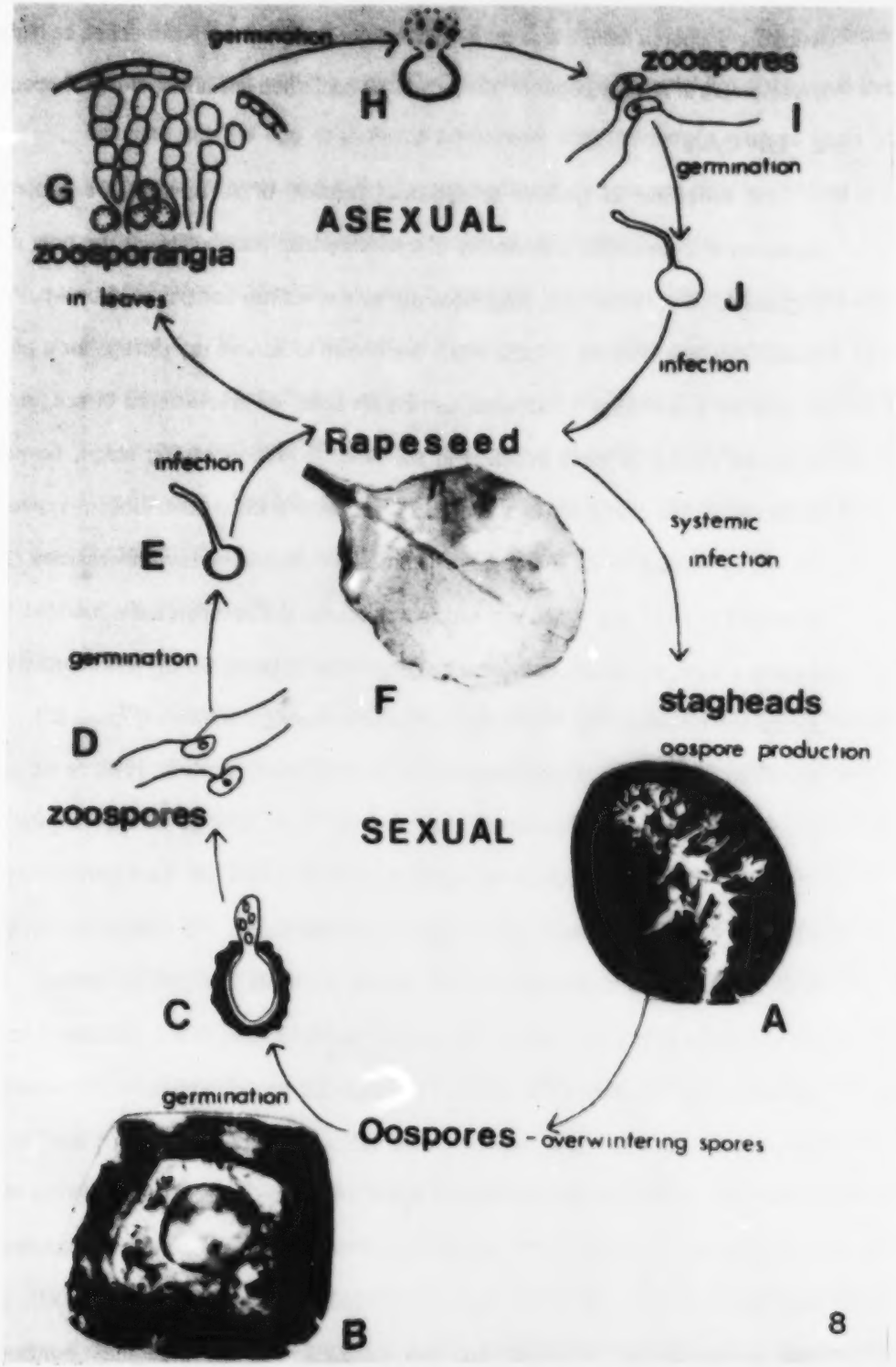


Figure 8. Life cycle of *Albugo candida*.

host (Figure 8F). The sporangia are disseminated by wind, by water or by other means onto the host surface. The sporangium, when it germinates, extrudes 4 to 12 zoospores into a sessile vesicle (Figure 8H). The zoospores (Figures 8I,J), on germination, infect new plants. Thus, the asexual cycle is repeated many times during the season. Sporangia are important only in the spread of the disease during the growing season, but play no importance in the survival of the pathogen during the non-crop season. The oospores, however, are important both for initiation of the disease as well as for the survival of the pathogen in the absence of the host.

1. **Survival**

a) Mycelium: It is believed that in perennial hosts such as horseradish, the mycelium is capable of overwintering in the infected crowns and lateral roots (57, 99, 266). Remaining dormant during the winter, it resumes activity and grows into the new shoots the host produces in the spring.

b) Sporangia: Sporangia of *A. bliti* attached to host tissues remain viable for 15 days at 3-10°C, whereas detached sporangia remain viable only for 24 h within this temperature range (147). At 30°C, viability is lost after 4 h when attached and after 2 h when detached from host tissues. In a recent study Lakra and Saharan (128) observed that sporangia of *A. candida* can survive for 4.5 days at 15°C on detached infected *B. juncea* leaves, but lose their viability after 18 h if separated and incubated without host tissues. However, sporangia can be stored for 105 days at -40°C as a dry powdered mass.

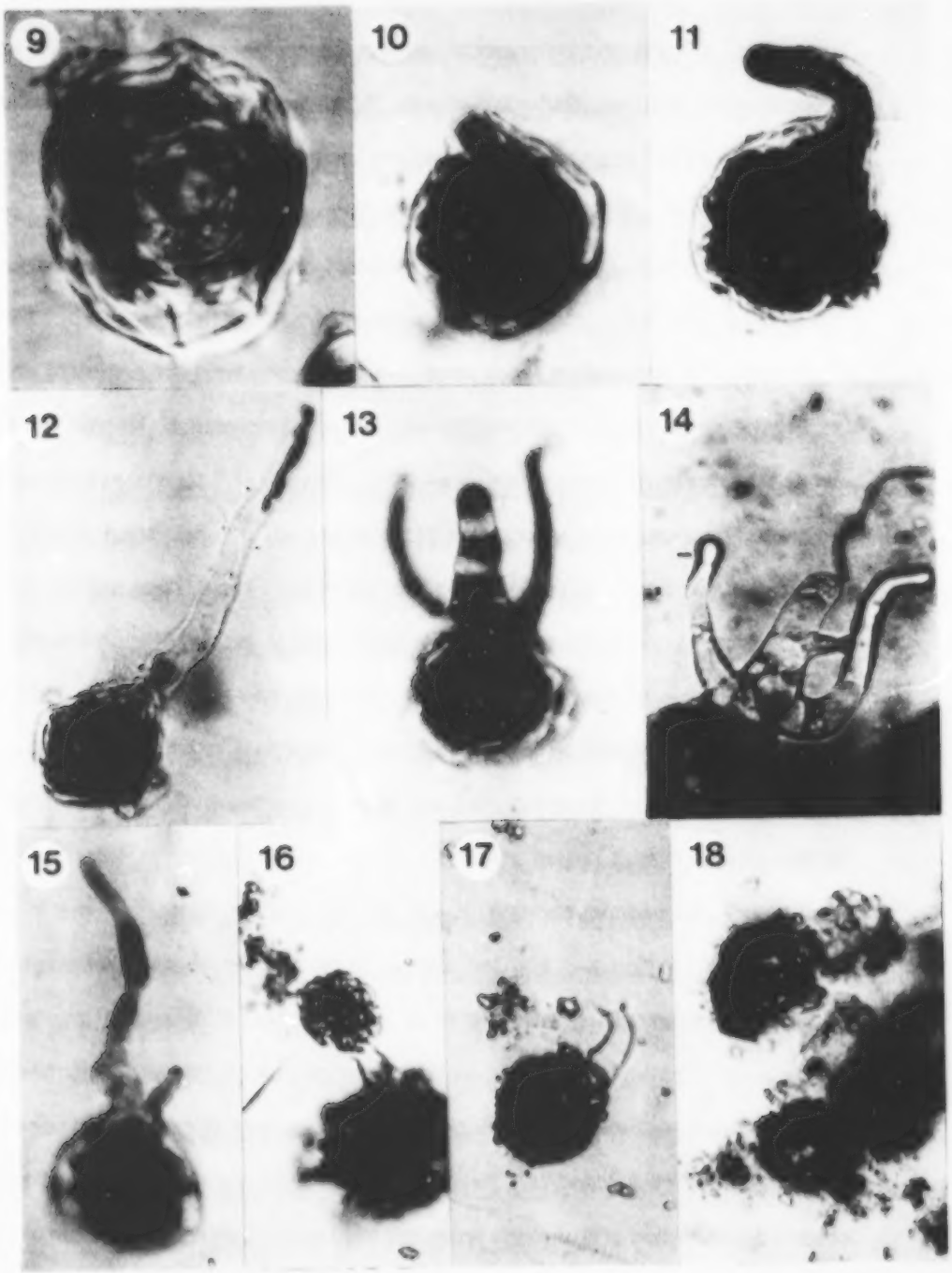
c. Oospores: Oospores are formed in the hypertrophied tissues (leaves, stems, inflorescences, pods, roots) of infected host plants. Overwintered oospores in infected plant debris in soil function as the source of primary inoculum of the pathogen (21, 23, 27, 99, 252, 266). Oospores have also been observed in naturally infected senesced leaves of *B. juncea* and *B. campestris.* var. Toria (P.R. Verma, unpublished data). Lakra and Saharan (127) estimated 8.75×10^5 oospores in one gram of hypertrophied cup-shaped leaves and 21.85×10^5 oospores in one gram of hypertrophied staghead

portions. Verma and Petrie (251) found that oospores can remain viable for over 20 years under dry storage conditions. According to Tewari and Skoropad (234), oospores have a highly differentiated, 5- layered cell wall; their greater longevity is probably due to the heavily fortified cell wall. Petrie (168) found more than 1500 oospores per gram of rapeseed seed and reported the possibility of survival and spread of the pathogen by means of oospores carried externally on seeds. However, information regarding production of oospores inside the seed and their possible importance in the survival of the pathogen are lacking.

2. **Spore germination**

a) Oospores: DeBary (41) first observed oospores of *Albugo* germinating via a sessile vesicle. Vanterpool (247) confirmed this and described a second mode of germination by means of a terminal vesicle; however, maximum germination was only 4% and its occurrence was unpredictable. Petrie and Verma (172) described a reliable and reproducible technique for inducing maximum germination of *A. candida* oospores. Verma and Petrie (251) found that maximum germination of *A. candida* oospores was 88% at 13°C from hypertrophied inflorescences of *B. campestris*. Oospores (Figure 9) germinated by the production of one or two simple or branched germ tubes (Figures 10-15), by the release of zoospores from vesicles formed at the ends of germ tubes (Figures 16,17), and by the release of zoospores from sessile vesicles (Figure 18). Germination by sessile vesicles was the most common. Verma and Bhowmik (258) observed that treatment of oospores with 200 ppm $KMnO_4$ for 10 minutes induces increased germination. Oospores do not appear to require any dormancy period. Verma and Petrie (251) were able to germinate 71% of the oospores within 2 weeks of collection from the field.

b) Sporangia: Sporangial germination in *A. candida* was studied by Prevost (177), Tulasne (244), Hoffmann (89), deBary (39), Melhus (144), Napper (152) and Endo and Linn (57). In 1911, Melhus reviewed the earlier work on sporangial germination. Prevost and deBary found that sporangial germination occurs via the production of



Figures 9-18. Germination of oospores of *Albugo candida* race 7. **Figure 9.** Mature oospore showing thick wall and well-developed central globule. **Figure 10.** Germ tube initial emerging from oospore and penetrating oogonial wall. **Figure 11.** Young germ tube. **Figure 12.** Long, less densely stained germ tube with some constriction at the exospore wall. **figures 13-15.** Branches arising from the main germ tube. **Figure 16.** Terminal vesicular mode of germination. **Figure 17.** An empty exit tube with a circular opening at its end. **Figure 18.** Sessile vesicular mode of germination (zoospores at top) (Can. J. Bot. 53: 836-842, 1975).

zoospores. Tulasne and Hoffmann stated that the sporangia germinate only by the production of germtubes. Harter and Weimer (79) stated that sporangia may germinate by the direct production of germ tubes but germination was more frequent via zoospores. Eberhardt (51), Melhus (144), and Napper (152) found that sporangia of *A. candida* germinate "invariably" by the production of zoospores; this was confirmed by the recent study of Lakra and Saharan (122) and Lakra *et al.* (130).

DeBary (39) and Melhus (144), reported that sporangia did not germinate above 25°C or below 0°C; the best germination was at lower temperatures. Napper (152) did not observe sporangial germination above 20°C. Melhus (144) suggested 10°C as the optimum temperature for sporangial germination, but Napper (152) found that germination takes place as readily at 1-18°C. Endo and Linn (57) reported the overall optimum temperature range for sporangial germination to be 15-20°C, with maximum germination occurring between 0 and 28°C. However, Lakra and Saharan (122) and Lakra *et al.* (130) observed >75% sporangial germination in *A. candida* at 12-14°C after 8 h incubation. Sporangia ceased to produce zoospores below 6°C and above 22°C. Sporangial germination started after 4 h and reached a maximum after 8 h of incubation. A quadratic equation, $Y = -103.16 + 26.99 x - 1.01 x^2$, where Y = % sporangial germination and x = temperature in °C was proposed to estimate the frequency of sporangial germination of *A. candida* from *B. juncea* at any known temperature. The variation in the cardinal temperatures for sporangial germination among different studies are probably due to the involvement of different host specific biological races of *A. candida*. Recently, Verma (P.R. Verma, unpublished data) observed germination of *A. candida* sporangia from naturally-infected *B. juncea* and *B. campestris* var. Toria leaves within one h at 13°C.

Melhus (144) and Holliday (90) reported that sporangial germination is not affected by light or darkness but recent studies by Lakra *et al.*(130) demonstrated that exposure to light of 150 $\mu EM^{-2}s^{-1}$ slightly delays sporangial germination in *A. candida* infecting *B. juncea*. Melhus (144) found that sporangia germinated as readily in a non-saturated as

in a saturated atmosphere. However, Lakra and Saharan (122) and Lakra *et al.* (130) found that a film of free water is essential for germination of sporangia. Melhus (144) and Napper (152) found that chilling and a reduction of 30% water content in sporangia is essential for germination. Lakra *et al.* (130) , however, states that it is not a prerequisite, since up to 75% of sporangia germinated without applying chilling or dehydration. According to Uppal (245), sporangia of *A. candida* require oxygen for germination. Takeshita (232) reported that sporangia of *A. candida* from horseradish germinated best at pH 4.5-7.5 at 10-20°C. Light did not affect germination. However, Endo and Linn (57) reported that sporangia of *A. candida* from horseradish require pH of 3.5-9.5 with an optimum of about 6.5; optimum temperature range was 15-20°C.

Only a few studies have been carried out on sporangial germination in *Albugo* species other than *A. candida*. Edie and Ho (54) demonstrated that although the sporangial germination in *A. ipomoeae-aquaticae* is nearly identical with that of other *Albugo* species with regard to the method of sporangial germination and host penetration, it requires a slightly higher germination temperature in the range of 12-30°C with an optimum of about 25°C. However, Saffeefulla and Thirumalachar (185) mentioned that sporangia germinated at 15°C but not at 24°C.

Sporangia of *A. ipomoeae-panduratae* germinate at 8-25°C (79) with an optimum of 12-14°C.

Sporangia of *A. tragopogonis* germinate at 4-35°C with an optimum range of 4-15°C. Encysted zoospores germinate best at 10°C (100). Sporangial germination of *A. tragopogonis* from *Senecio squalidus* occurs at 5-15°C, with an optimum of 10-15°C. Very little germination occurs at 20°C (268, 269).

Sporangia of *A. bliti* germinate at 2-25°C, with an optimum of 18°C (147). Chilling of sporangia increases percentage of germination. Mature sporangia from just-opened pustules, or those naturally detached, germinated best.

Sporangia of *A. occidentalis* germinate at 2-25°C with an optimum near 12°C (180). Light, water loss from sporangia, and pH have little effect on sporangial

germination.

3. Infection

In all species of *Albugo* , oospores are the primary source of inoculum (21, 23, 27, 81, 252, 266). In perennial hosts like horseradish, the mycelium persists in the crowns and occasionally in lateral roots (99). Secondary spread is by means of sporangia, which are readily carried short distances by splashed water droplets or, to a certain degree, by air currents. Moisture on the host surface is essential for germination of sporangia and infection by zoospores. The most likely primary infection sites are the emerging cotyledons (252). Zoospores derived from oospores are capable of causing infection (252), but no evidence exists of infection arising directly by germ tubes derived from oospores (251). Sporangia and oospores germinate to give biflagellate zoospores, about 4-12 per sporangium and 40-60 per oospore (251). Raabe and Pound (180) reported a persistent terminal bead on one flagellum of *A. occidentalis* zoospores. Whipps and Cooke (270) observed the appearance and subsequent disappearance of flagellar beads during zoospore differentiation, and the absorption of beads by encysting zoospores of *A. tragopogonis*. After swimming for a time, a zoospore encysts and forms a germ tube, which enters the host through a stoma (252). Germ tubes enter the stomata of resistant hosts as readily as those of susceptible hosts (252). In the former, mycelial growth of the pathogen ceases in the substomatal chamber and a marked encapsulation forms around each single haustorium (252). In the congenial or susceptible host, the mycelium advances intercelluiarly with the production of haustoria (134, 135, 152, 252, 266). According to Liu and Rimmer (134), the host genotype, inoculum concentration, and incubation temperature, as well as the interactions among these factors had significant effects on white rust infection. The day/night temperature of 22/17^{o}C was more favourable to fungal growth than that of 15/10^{o}C. Infection levels increased with the concentration of inoculum on each line/cultivar at both the two-leaf and five-leaf growth stages of *B. napus* and *B. rapa*. While studying the histopathology of compatibility and incompatibility between oilseed

rape and *Albugo candida* race 7, Liu *et al.* (135) observed haustorium formation in the palisade mesophyll cells adjacent to the substomatal chambers 8 h after inoculation. In the resistant cv., most primary hyphae produced one haustorium. Necrosis of the invaded host cells was first observed 12 h after inoculation followed by cessation of fungal growth. The death of host cells was largely restricted to the penetration site, the adjacent non-penetrated cells remained apparently unaffected. In the susceptible hosts, necrosis of infected cells occurred only infrequently, and hyphal growth continued unabated, resulting in mycelial ramifications into the mesophyll. Numerous haustoria per infection site were produced. The presence of as many as 14 haustoria in a single cell in "green island" tissue of artificially infected *B. juncea* cotyledons have been reported (252).

4. Disease Development

Severity and incidence of white rust varies from year to year on different crops. Factors affecting the initiation and development of the disease have not been studied in detail.

On spinach, sporangial production is profuse at low temperatures, but oospore production is very abundant at higher temperatures (180). On water spinach in Hong Kong during the main growing season (May to October), temperatures of 24-29°C with heavy dew formation is ideal for infection and disease development (54). On sunflower in Australia, white rust development is greatest between 20 and 25°C (100).

Sempio (201-203) reported that the optimum temperature for the development of white rust on potted radish plants was 16-18°C, with a range of 12-21°C. The disease did not develop below 6-7°C or above 28-29°C. Relative humidity of 60-80% was more favourable than a saturated environment. CO_2 between 70 and 80 mm Hg (9.6-11%) inhibited the disease. The fungus was highly sensitive to the effect of ultraviolet light; the optimum dose for inhibition of disease was 25-30 minutes. White rust of horseradish is most favoured at 15-20°C (232).

On Amaranthus species, ideal conditions for the spread of white rust around Delhi

(India) occurs from October to the end of February. During this period, warm days with low humidity facilitate desiccation of sporangia, while cool temperatures and heavy dew deposition at night provides optimum conditions for sporangial germination and infection (147).

White rust development in field on *B. juncea* in relation to environmental conditions in India has been studied (123, 131, 187, 189, 259). White rust pustules increase at a faster rate when the mean temperature is 11.5-12.5°C, mean RH is >75%, cloudy weather is coupled with precipitation and wind velocity is 2.6 km/h. Delays in sowing until after October 19 increases disease intensity. Late planting (3rd week of October or first week of November) causes high incidence (10-43%) and severity (13-32%) of stagheads in toria and mustard (112, 115). These authors also reported that 2 to 6 h of sunshine per day, concomitant with a mean minimum temperature of 6-10°C, a mean maximum temperature of 21-25°C and rainfall up to 160 mm during the flowering - cum -pod formation stages is favourable for severe development of disease in toria and mustard (114, 115). In their two years of field experiments in India, Verma and Bhowmik (259) showed that a mean temperature of <16°C and a mean R.H. of >60% were necessary for white rust development in mustard. Infection rate was the highest (0.574) at a mean temperature of 14.72°C and a R.H. of 73.25%; infection rate decreased with the increase in mean temperature and decrease in mean R.H. Rainfall of 9 inches coincided with the highest infection rate. Duration of sunshine hours did not have any marked effect, although prolonged periods coincided with poor disease development. In a laboratory study Verma *et al.* (256) found maximum production of pustules on medium aged detached leaves of *B. campestris* cv. Torch incubated at 18.5°C and inoculated with race 7 of *A. candida*.

4. PHYSIOLOGIC SPECIALIZATION

Physiologic specialization has long been known in *A. candida*. Eberhardt (51) recognized two specialized groupings of *Albugo*, one attacking *Capsella, Lepidium* and *Arabis*, and the other attacking *Brassica, Sinapis* and *Diplotaxis*. He was, however,

hesitant to use the phrase "biological forms" for this fungus. Later, Melhus (144) also suggested the existence of specialization in *A. candida*. Pape and Rabbas (160) demonstrated that the fungus on *Capsella bursa-pastoris* should be considered a distinct form. Pfister (173) found several biological forms in *A. tragopogonis*. Taubenhaus (233) and Ciferri (28) reported that distinct races occurred on *Ipomoea batatas* and on *Ipomoea* spp. in the USA and West Indies. Ciferri (28) divided *A. ipomoeae-panduratae* into two biologic species. Savulescu and Rayas (197) distinguished eight morphological forms within *A. candida*. Later, Savulescu (195) made 10 varieties of *A. candida* based on host specialization and morphology. Hiura (87) distinguished three biologic forms of *A. candida*, the first on *Raphanus sativus*, the second on *B. juncea* and the third on *B. campestris* sp. *chinensis*. Napper (152) described 20 races of *A. candida* in Britain. Togashi and Shibasaki (242) found that sporangia of Albugo from *Brassica* and *Raphanus* were 20 x 18 µm in size, while those from *Cardamine, Capsella, Draba,* and *Arabis* measured 15.5 x 14.5 µm; these were classified as macrospora and microspora, respectively. Results of this Japanese study suggested that five distinct biological forms of *Albugo* were present. Subsequently, Ito and Tokunago (96) elevated the forms with the larger spores to the rank of the species *Albugo macrospora* (Togashi) Ito. Biga (13) recognized two morphological taxa: *A. candida macrospora* and *A. candida microspora,* as proposed by Togashi and Shibaskaki (242), but renamed them *A. candida microspora* and *A. candida candida*, respectively. On the basis of conidial measurements from 63 species, Biga (13) reported that *A. candida microspora* (15-17.5 µm-diameter) is restricted to *Armoracia, Brassica, Erucastrum, Raphanus*, and *Rapistrum*, whereas *A. candida candida* (12.5-15 µm-diameter) has a wide range of cruciferous hosts. In addition he identified two intraspecific taxa of *A. ipomoeae-panduratae* and five of *A. tragopogonis*. *Albugo tragopogonis* was considered by Savulescu (195) to have two varieties differing in spore shape each of which included morphological forms differing in size. Biga (13) distinguished 5 morphological varieties in this species. Endo and Linn (57) reported one race of *Albugo on Armoracia rusticana*.

It is clear that each of the above authors was hesitant in describing specialized races of *A. candida*. Pound and Williams (176) identified 6 races of *A. candida*; race 1 from *Raphanus sativus* var. Early Scarlet Globe, race 2 from *B. juncea* var. Southern Giant Curled, race 3 from *Armoracia rusticana* var. common, race 4 from *Capsella bursa-pastoris*, race 5 from *Sisymbrium officinale*, and race 6 from *Rorippa islandica*. Race 7 from *B. campestris* Turnip or Polish rapeseed and race 8 from *B. nigra* were added by Verma *et al.*, (234) and Delwiche and Williams (44), respectively.

In Russia, Novotel'nova (156), while analyzing intraspecific taxa, established that the *A. candida* species consists of separate morphological specialized forms confined to a particular range of host plants, i.e., to plants of certain species or groups of genera and species. Within the morphological forms, races can be differentiated, while within heterogeneous populations both races and forms can be differentiated. Geographical and climatic conditions leave their distinguishing mark on the processes of form development so that populations of the fungus encountered by investigators in different countries were not identical. Novotel'nova and Minasyan (157) and Burdyukova (20) studied the biology of *A. candida* and *A. tragopogonis* in Russia and conducted an in-depth study of the extent of specialization in *A. candida*.

In India, Singh and Bhardwaj (211) tested 12 *Brassica* species and identified 9 races from four hosts, viz., *B. juncea, B. campestris* var. Toria, *B. campestris* var. Brown Sarson and *B. campestris* var. pekinensis. Recently, Lakra and Saharan (124) identified five races of *A. candida* on the basis of their reaction on a set of 16 host differentials. They identified two distinct races from *B. juncea* which are different from previous records. One (race 2), attacks *B. nigra, B. juncea* and *B. campestris* var. Brown Sarson, and the other (race 3), infects only *B. juncea* and *B. campestris* var. Toria. The concept of races in *A. candida*, as proposed by Pound and Williams (176), was based on species relationships. Recent studies have, however, clearly demonstrated that cultivars of *Brassica* crops must be included in a set of host differentials to distinguish isolates of the pathogen within a presently accepted race (20, 174). There is an urgent need to standardize host

differentials keeping in mind the homogeneity and purity of species and varieties. A detailed study regarding identification of biological races of *A. candida* occurring on different *Brassica* crops in India using differential hosts described by Pound and Williams (176) is still lacking. Using North American races 2 and 7 from *B. juncea* and *B. campestris*, respectively, Petrie (170) recently screened accessions of several *Brassica* species including *B. campestris* var. Yellow Sarson, *B. campestris* var. Brown Sarson, *B. campestris* var. Toria, and *B. juncea* from India: both Yellow and Brown Sarson were equally highly susceptible to both races, Toria only to race 7 and *B. juncea* only to race 2. A detailed study is needed to determine whether races of *A. candida* attacking *B. juncea* and several *B. campestris* crops in India are similar to races 2 and 7 from Canada and the U.S.A. Using all the differential hosts of Pound and Williams (176), Verma (250) reported that in India, the *A. candida* race attacking *B. juncea* does not appear to be distinct from the race attacking *B. campestris* var. Toria; observations were based on preliminary cross inoculation studies in the greenhouse.

5. THE GENETICS OF HOST-PARASITE INTERACTION

Studies on the genetics of host-parasite interactions in white rust diseases has focussed on the level of specificity among races of pathogens and genotypes of related host species. Even within the confines of race-cultivar specificity, the studies have been one-sided in that no genetic information has been generated on *Albugo*, the causal organism. Interest in such studies was stimulated by Hougas *et al.* (92), who studied the genetic control of resistance in white rust of horseradish. The exhaustive work of Williams and Pound (176) clearly demonstrated that resistance to white rust in radish cv. China Rose Winter (CRW) and Round Black Spanish (RBS) was controlled by a single dominant gene. Histological studies revealed that resistance in CRW is manifested as a hypersensitive reaction, which may be modified to a sporulating tolerant reaction by environmentally controlled minor genes. Humaydan and Williams (95), while studying the inheritance of resistance in radish to *A. candida* race 1, changed the gene designation "R" into the more descriptive symbol "AC-1", derived from the initials and race number of *A.*

candida. The resistance to *A. candida* race 1 in *Raphanus sativus* cv. Caudatus is controlled by a single dominant gene, AC-1. The resistance gene AC-1 and the gene Pi, controlling pink pigmentation, were found to be linked with a recombination value of 3.28%. Bonnet (18) found that white rust resistance in radish variety "Rubiso 2" is also controlled by one dominant gene.

Among *Brassica* species, monogenic dominant resistance to *A. candida* race 2 has been found in *B. nigra, B. campestris, B. carinata*, and *B. juncea* (42, 52, 239). A single dominant gene,AC-2, controlling resistance to *A. candida* race 2 in *B. nigra* was identified by Delwiche and Williams in 1981 (45). In a study to select quantitatively inherited resistance to *A. candida* race 2 in *B. campestris*, CGS-1, Edwards and Williams (55) found that variability in reaction to *A. candida* race 2 among susceptible *B. campestris* strain PHW - Aaa-1 was due to quantitative genetic regulation, and suggested that rapid progress in resistance breeding could be made via mass selection when starting with a susceptible base population.

Canadian cultivars of *B. napus* are resistant to white rust, but many cultivars of this species grown in China are susceptible (58). The inheritance of white rust resistance in *B. napus* cv. Regent is conditioned by independent dominant genes at three loci, designated as AC-7-1, AC-7-2 and AC-7-3. Resistance is conferred by dominance at any one of these loci, while plants with recessive alleles at all loci are susceptible. Results of a recent study by Verma and Bhowmik (260) are in part agreement with those of Fan *et al.* (58) and suggest that resistance of BN-Sel (*B. napus*) to the *B. juncea* pathotype of *A. candida* found in India is conditioned by dominant duplicate genes.

In a recent study of inheritance of resistance to *A. candida* race 2 in mustard, Tiwari *et al.* (240) found that resistance is dominant, monogenic, controlled by nuclear genes and is easily transferred to adapted susceptible genotypes via backcrossing. In a study evaluating performance of fifteen advanced generations (F_6) progenies of 2 interspecific crosses of *B. juncea* and *B. carinata* against white rust, Singh *et al.* (213) showed significant differences among the progenies, and that all the hybrid progenies gave

resistant reaction. In a later study, five interspecific crosses between *B. juncea* and *B. carinata* revealed that the dominant gene which conferred resistance to white rust is located in "c" genome of *B. oleracea*, a proginator of *B. carinata* (212). Williams and Hill (277), and Edwards and Williams (56) have opened unusual potential for resolving many problems relating to host-parasite interactions and breeding for disease resistance through development of rapid-cycling *Brassica* populations. Their preliminary studies demonstrated considerable isozyme variations among individuals in populations which, when inoculated with several pathogens, showed a wide range of plant-to-plant variation in the levels of resistance and susceptibility. This will assist plant breeders in developing cultivars with genetic resistance to plant diseases. These authors and Hill *et al.* (84) are currently constructing gene pools of both major and minor genes for resistance to various crucifer pathogens which will be of immense value to plant breeders seeking sources of resistance.

6. SOURCES OF RESISTANCE

Considerable efforts have been made to evaluate cultivar resistance to *Albugo* in cruciferous crops. Williams and Pound (275) reported that resistance in radish cvs. China Rose Winter (CRW) and Round Black Spanish (RBS) is governed by a single dominant gene. Humaydan and Williams (95) identified a single dominant gene, AC-1, in radish cv. Caudatus. Bonnet (18) reported that radish cv. Rubiso 2 contains a single dominant gene.

Delwiche and Williams (42) reported that all accessions of *B. napus* and most cultivars of *B. oleracea* are resistant to *A. candida*. Resistance in *B. napus* cv. Regent is governed by three genes, AC-7-1, AC-7-2, and AC-7-3 (58). Several accessions of B. juncea, B. campestris, var. Yellow Sarson, and B. campestris var. Brown Sarson are resistant to A. candida (52, 102, 114, 116, 136, 162, 187, 189, 220).

Canadian breeders and plant pathologists in 1980 licensed *B. campestris* cv. Tobin, which was specifically bred to be highly resistant to race 7 of *A. candida*. Tobin was also found to be resistant to white rust in India.

In Texas, U.S.A., some resistance has also been reported in certain varieties of spinach against *A. occidentalis* (33).

7. FINE STRUCTURES

Electron microscopy, particularly when used in association with physiological, biochemical and genetic studies, provides valuable information on the complex relationships which exist between host and pathogen.

a) Haustoria: The fine structures of *A. candida* were studied by Berlin and Bowen (10), Davison (38) and Coffey (29). The small stalked, capitate haustoria of *Albugo* are connected to the much larger haustorial mother cell by a slender cylindrical neck. Haustoria contain mitochondria with tubular cristae, ribosomes and occasional cisternae of rough endoplasmic reticulum. Nuclei and perinuclear dictyosomes, although present in the mother cells, are absent in the haustoria. The fungal plasma membrane and cell wall are continuous from an intercellular hypha to the haustorium except that there is no evidence of a fungal cell wall around a portion of the haustorial stalk proximal to the haustorial head. In the host mesophyll cell, the haustorium is invariably surrounded by host plasma membrane and/or a thin layer of host cytoplasm. The host cell wall invaginates at the point of haustorial penetration to form a short sheath around the penetration site, but the host cell wall is absent from rest of the haustorium. A collar consisting of fibrillar material is commonly found around the proximal portion of the neck. An electron-opaque encapsulation lies between the haustorium and the host plasma membrane and extends into the penetration region between the sheath and the fungal cell wall. An electron-opaque sheath surrounds the thin wall of the haustorial body but is absent from the neck region. A series of tubules is continuous with the invaginated host plasma membrane which surrounds the haustorial body. These tubules contain an electron-dense core similar in appearance to, and continuous with, the sheath matrix. Host dictyosomes and their secretory vesicles are not involved in formation of the haustorial sheath. A constant feature of the haustorial apparatus is the association of flattened cisternae of host endoplasmic reticulum with the distal portion of the haustorial neck. Woods and Gay (279) provided evidence for a neckband delimiting structural and physiological regions of the host plasma membrane associated with haustoria of *A. candida*. Coffey (30) demonstrated cytochemi-

cal specialization at the haustorial interface of *A. candida*.

b. Sporangia: Paramural bodies are formed in sporangia of *A. candida* solely by elaborations of the plasma membrane. Two major forms have been recognized, one consisting of invaginations of the plasma membrane projecting into the cytoplasm and the other appearing like pockets containing vesicles and tubules. The first may be the basic form of the paramural body. In sporangia, the paramural bodies break away from the plasma membrane and undergo autodigestion. In vegetative hyphae, the tubules and lamellae of paramural bodies break up into vesicles and are finally sequestered into the cell wall (105-107). The surface layer of the cell wall of the sporangia and sporangiophores of *A. candida* is composed of a series of lamellae. Evidence from freeze-fracture, freeze-etch, and single-stage replicas demonstrated that the lamellae are bilayered, an organization associated with the presence of lipids. This multilamellate layer on the surface of the cell wall facilitates air dispersal and protects the sporangia from desiccation (235).

In *Albugo* sporangia are produced in basipetal chains at the apices of sporangiophores and are released by the dissolution of the septa that delimit them. Hughes (93) suggested that sporangiophores of *Albugo* produce sporangial chains by percurrent proliferation and that they are "apparently the morphological equivalents of annellophores (annellides)" (94). A sporangial initial buds out from a fixed locus at the tip of the sporangiophore. After reaching a certain size, it is delimited by a basal septum and converted into a sporangium. A new initial grows out from the sporogenous locus, pushing the newly formed sporangium upward. By repetition of this process, a basipital chain of sporangia is formed. Both layers of the sporangiophore wall grow out and take part in forming the sporangial wall. In conidium ontogeny this mode of development is called holoblastic. During sporangial formation in *A. candida* the sporangiophores do not increase in length; however, abnormally long sporangiophores are sometimes seen among the smaller, regular ones. There are no annellations on the sporangiophore surface and no increase in the thickness of the sporangiophore wall at its apex. Thus, none of the characteristics that have been shown to be associated with percurrent proliferation are present during the development of

sporangia in *Albugo* (107). In maturing sporangia a burst of activity was observed by Khan (105, 106). Even after formation of sporangia, the numbers of mitochondria and the amounts of endoplasmic reticulum increase. Perinuclear vesicles and smooth surface cisternae differentiate into well developed Golgi apparatuses, which remain secretory until complete maturation of sporangia. Maturing sporangia have autophagic vacuoles containing various cell organelles. Nuclear degeneration and mitosis proceed simultaneously. All activities decline towards the end of sporangial maturation. Golgi dictyosomes become quiescent and the numbers of mitochondria and amounts of endoplasmic reticulum decrease. There is a three-fold increase in the thickness of the sporangial wall during maturation.

c. Oospores: The structure and development of oospores of *A. candida* in the stagheads on rapeseed (*B. campestris*) were investigated by light microscopy, transmission electron microscopy of ultrathin sections and scanning electron microscopy (234). Development of an oospore was similar to that in *Pythium* (234). A reaction zone forms on the oogonial wall at the point of contact by the fertilization tube of the antheridium. The oospore has a highly differentiated, five-layered cell wall. The periplasm appears to play an active role in the deposition of the oospore cell wall. The contents of the periplasm do not disappear after maturation of the oospore; instead, they form a persistent material between it and the oogonial wall. Hence, functionally, the oospore wall complex has two additional layers which may contribute to the longevity of the oospore.

In a histochemical study of cytoplasmic changes during wall layer formation on the oospore of *A. candida*, Kauer *et al.* (103) reported that the young multinucleate oogonium is double-walled. The oospore nuclei are large and prominent and have an outer shell or sheath of proteinaceous material surrounding a central core of nucleoplasm. The first wall of the fertilized oospore is laid at the interphase of the periplasm and the ooplasm. Subsequent wall layers are formed both on the inner and outer side of the first oospore wall. The second oospore wall is formed just internal to the first one. The third wall of the oospore is formed external to the first one and appears ridged. The last wall to be formed

is the innermost one which completely surrounds the central ooplasm. This wall layer is callosic in nature.

8. BIOCHEMISTRY OF HOST-PARASITE INTERACTION

Biochemical studies of the growth and survival of a pathogen and of the changes it induces in its host can ultimately lead to a better understanding of epidemiology, disease development and control. With few exceptions, such studies on white rust lag far behind those for diseases caused by other major groups of biotrophs. Ideal prerequisites for meaningful studies of the biochemistry of host-parasite interaction are a) a clear understanding of the genetic control of virulence and avirulence in the parasite and of susceptibility and resistance in the host, b) precise histological and cytological descriptions of spore germination, infection and the establishment and development of the infection, and c) the availability of methods for growing the parasite alone and in combination with its host under controlled conditions. Unfortunately, these criteria have not been fully satisfied for any white rust disease.

a. Carbohydrate metabolism and respiration: A characteristic feature of the infection of plant tissues by Uredinales and Erysiphaceae is a two to four fold increase in the rate of respiration (83, 200). Most evidence suggested that in these cases host respiration is shifted from a system that is predominantly channeled through glycolysis and the Krebs cycle to one that is dependent on the pentose phosphate pathway (32). Increases in the activities of metabolites produced in the Krebs cycle have also been reported, but the relative contributions of this and the pentose phosphate pathways are not clear. A number of reports indicate that the respiration rates of tissues infected by members of the Albuginaceae also rise dramatically (14, 15, 276). However, the limited evidence so far suggests that such increases do not reflect any significant change in the pentose phosphate pathway.

Radish cotyledons infected with *A. candida* increase respiration three-fold, but the c_1/c_6 ratio, together with evidence from inhibition and feeding experiments, suggests that this was due to stimulation of existing pathways and did not involve the pentose phosphate

pathway (14, 15, 276). Long and Cooke (136) suggested that host-fungus movement of carbohydrates in *Albugo - Senecio squalidus* system is maintained by hydrolysis of host sucrose and uptake of hexoses, followed by accumulation of trehalose within the mycelium and spores. Trehalose was synthesized within pustules by the fungus but no acyclic polyols were found. Accumulation of hexoses around pustules together with increased hydrolysis of exogenous sucrose by pustular material indicated increased invertase activity within infected tissues. Accumulation of dark-fixed carbon compounds in white rust pustules of *Senecio squalidus* infected with *A. tragopogonis* has been reported (238).

b. RNA content: In *Ipomoea* white rust there was greater reduction in the RNA content of infected tissues than in the healthy, adjacent tissues (146).

c. Photosynthesis: Black *et al.* (14) used infrared CO_2 analysis to demonstrate that a decline in the photosynthetic rate of cotyledons of radish infected with *A. candida* preceded the rise in respiration rate reported by Williams and Pound (276). In another study Harding *et al.* (77) examined the pattern of pigment retention during green island development following infection of *B. juncea* cotyledons with *A. candida*, and studied the photosynthetic capacity of green island tissue and the ultrastructure of chloroplasts within them. They found that labelled glycine $2\text{-}^{14}C$ was incorporated into chlorophylls a and b in both infected and non-infected tissue. Both infected and non-infected tissue fixed $^{14}CO_2$ in the light, but 4 days after infection green islands fixed five times more $^{14}CO_2$ in the light than did non-infected tissue. Photosynthesis per mole of chlorophyll fell at the same rate in green island as in non-infected tissue. The maintenance of chlorophyll and continued photosynthetic activity in green island tissue was paralleled by delayed breakdown of chloroplasts. Electron microscopy showed that these retained their structural integrity longer in green islands than in non-infected tissue. The authors drew attention to the similarity in *B. juncea* between green islands and tissues treated with kinetin.

Studies of the effects of white rusts and related pathogens on photosynthesis have been superficial, so meaningful comparisons with the rusts and powdery mildews are difficult. Extensive research has indicated that the overall activity of photosynthetic

pathways declines in leaves infected by rusts and powdery mildews, and is accompanied by a decrease in chlorophyll content of the tissue (32, 36). Around the sites of infection, however, green islands of chlorophyll are retained and within them photosynthesis continues. Thus the green islands are regions within which synthetic processes are maintained and the movement of metabolites from host to parasite may continue after the rest of the leaf has become non-functional. Enhanced fixation of CO_2 in the dark also occurs in green islands, but this appears to be due to the ability of the fungus itself to dark fix. The similarity between green islands and the sites of exogenous application of cytokinins to plant tissues has been widely noted, although there is no clear evidence that these or other plant growth substances are actually responsible for the green island phenomenon.

d. Accumulation of metabolites: Long *et al.* (132) demonstrated that infection of *S. squalidus* by *A. tragopogonis* results in an increase in the activity of acid invertase at the infection site. They concluded that sucrose from the host is first hydrolyzed to glucose and fructose and then absorbed by the parasite. They also suggested that invertase may play a key role in the provision of substrate for the accumulation of starch at infection sites: where there is a surplus of soluble carbohydrate, particularly sucrose, hydrolysis by invertase might provide hexose for starch synthesis within chloroplasts. Invertase may thus mediate a system by which the excess soluble carbohydrate at infection sites is converted to osmotically inactive polysaccharides. In contrast, Whipps and Cooke (271) suggested that starch does not accumulate in *S. squalidus* infected by *A. tragopogonis*. Further research is required to resolve this conflict.

Dhingra *et al.* (48) found decreased amounts of free protein, total protein and total phenolic compounds in floral parts and floral axes of *B. campestris* infected with white rust. Dhawan *et al.* (47) correlated resistance of *B. juncea* cv. RC-781 with higher concentrations of phenols when compared with the susceptible cvs. Prakash and RH-30, where greater amounts of sugars were present.

Purohit *et al.*(178) observed gradual increases in peroxidase and IAA-oxidase activity

with increased severity of white rust (*A. bliti*) infection on leaves of *Achyranthes aspera*. A decrease in total and o-dihydroxy phenols with increased poly-phenol-oxidase activity was observed in infected leaves when compared with healthy tissue. A gradual increase in enzymatic activity with loss of phenolics shows a state of high catabolism induced by pathogenesis. Singh *et al.* (217) demonstrated that cellulase, endo-PMG and endo-PG were produced in *B. juncea* leaves infected with *A. candida*. According to Maheshwari and Chaturvedi (140) the swelling and disruption of subcellular particles rich in lysomal acid hydrolases was produced by acid phosphatase activity centered primarily in the infected tissues of *B. juncea*. Acid phosphatase activity in antheridia, oogonia and oospores of *A. candida* indicates that the enzyme plays a role in the synthesis of fungal organs.

e. Growth substances: Infection of host plants with *Albugo* causes hyperplasia and hypertrophy of leaf, stem and floral parts. Kiermayer (108) found that these symptoms are produced in plants infected with *A. candida* due to the production of indolacetic acid (IAA). Hirata (85, 86) found that infection with *A. candida* causes an initial increase in diffusible auxin in diseased stems and leaf sections, followed by a decrease before maximum development of the galls. The auxins in healthy and *Albugo*-infected inflorescences of *B. napus* have now been identified and estimated quantitatively by Srivastava *et al.* (219). Malformed *B. napus* inflorescences produce IAA, IAN, accelerator L, and an ether-insoluble growth substance designated as A.

Kumari *et al.* (119) and Lal *et al.* (132) studied the quantitative and qualitative changes in the amino acid contents of diseased (hypertrophied) and healthy tissues of mustard and radish. Kumari *et al.* (119) suggested that the amino acid contents are greatly changed because of disruption in the metabolism of plant organs under the influence of a pathogen. The infection causes the breakdown of plant proteins, releasing small quantities of tryptophan, which reacts with endogenic phenolic acid to produce IAA which is responsible for hypertrophied growth.

It is clear from the above that there is an urgent need to gather basic information concerning the effects of white rust on respiration, photosynthesis, accumulation and transfer

of carbohydrates, production of growth regulators, and the role of phenolics and other growth substances in infected host tissues.

9. TECHNIQUES

a. Axenic culture: The growth of *Albugo* in callus cultures of *Ipomoea* has been obtained by Singh (214). He observed mycelium, haustoria, sporangiophores, sporangia and oospores on the callus tissue. Repeated subculturing at 40-day intervals was required for good fungal growth. The various globules of the callus which were repeatedly cultured could be distinguished into fungus free globules, globules with fungus and oospores (215).

b. Sporangial viability test: A fluorescent microscopy technique to test sporangial viability was suggested by Vyalyka and Lanetsku (262). This method provides information on the number of viable sporangia of *Albugo* in a population within minutes. Sporangia are stained with a 0.01% aqueous solution of acridine orange for 5-10 min and viewed under a fluorescence microscope. The dead sporangia will fluoresce green.

c. Sporangial preservation: Freezing (280) has been used to maintain the white rust fungus on living plants. In 1958, O'Brien and Webb (158) suggested that *Albugo occidentalis* can be preserved for 5-6 months by storing infected spinach leaves at -10°C. According to Lakra and Saharan (128), sporangia of *A. candida* from *B. juncea* survive for 4.5 days at 15°C on detached infected leaves, but lose their viability after 18 h if they are stored as a dry powdered mass. However, sporangia can be stored as a dry powdered mass for 105 days at -40°C.

d. Inoculation applicator: A micro ULVA controlled droplet applicator was developed as a tool for inoculating *A. candida* (race 2) in the field and greenhouse by Perry and Williams (164). The severity of the disease is greater in the field or greenhouse when large spray droplets (60-70 µM) are used instead of small droplets (30-40 µM). Since relatively small volumes of inoculum are required, this inoculator is a potentially valuable tool for field use.

e. Components of partial resistance: Partial resistance may be the result of low infection frequency, low spore production, a long latent period, and/or a short infectious period. Fox and Williams (61) stated that spore production by *A. candida* on *Brassica*

campestris is highly correlated (r = 0.93) with a visual white rust interaction phenotype (IP) rating scale. Plants are rated from 0-9 according to the amount of leaf necrosis or area covered by white rust pustules. Means of spore production on plants rated as 1,3,5, and 7 are significantly different (P = 0.05) from each other. These results support the use of a visual scale to select for components of partial resistance to the white rust disease. Black and Dainello (16) made comparisons of percent leaf area with white rust lesions and two other methods for evaluating partial resistance to *A. occidentalis* in spinach. Lakra and Saharan (121), however, using length of incubation period to determine level of resistance in several *B. juncea* cultivars found that the decision between compatible (susceptible) and incompatible (resistant) reaction of *Albugo-Brassica juncea* system is made within 80 h of inoculation. In a histopathological study, Liu *et al.* (135) showed that the earliest event distinguishing a compatible from an incompatible interaction occurs after formation of the first haustorium and that resistance is not manifested until the host mesophyll cell had come into contact with the first haustorium.

f. White rust as a weed control tool: Disease induced by *A. candida* has a significant effect on the survival and reproduction output of *Capsella bursa-pastoris* (Shepherd's purse), a weed plant. The timing of infection is very important in determining the ultimate effect of the disease. Systemic (primary) infection of seedlings causes a high degree of mortality (88%) prior to reproductive maturity. Localized or systemic secondary infection does not affect survival (2).

In North America, Hartmann and Watson (80) suggested using the white rust fungus *A. tragopogonis* to control common ragweed (*Ambrosia artemisiifolia*). When the plants are inoculated at the two leaf stage, pollen production is reduced by 99%, seed production by 98% and top weight by 79% when infection is systemic. A convenient method of inoculation was described.

g. Oospore germination: Germination of oospores of *A. candida* in the laboratory was first reported in 1866 (41). Petrie and Verma (172) developed a reliable technique by which 70% oospore germination was achieved. Later Verma and Petrie (251) refined this and

developed two more techniques to get even better results. In all three techniques, hypertrophied tissues are finely ground with a mortar and pestle, and the grindings are screened through a 60-mesh sieve to give a brown powder consisting largely of oospores. The oospore were germinated by incubating them on moist filter paper for 21 days at 10-15°C, by slowly leaching them for 15 days or more on sintered glass filters, or by washing them for a few days on a rotary shaker followed by a day of still-culture. Recent paper by Verma and Bhowmik (258) suggests that pre-treatment of oospores with $KMnO_4$ gives better germination.

h. Disease scoring scale: Pound and Williams (176) reported a procedure to assess symptoms of white rust on mature cruciferous plants. This procedure has since been modified by different workers. Mayee and Datar (142) suggested the use of 0-9 scale based on growth stages of the crop for leaf as well as staghead infections. To assess leaf phase and staghead phase infections separately, and/or in combination, a 0-5 scoring scale has been prepared by Lakra and Saharan (131).

A descriptive and pictorial white rust assessment scale for sunflower was proposed by Siddiqui *et al.* (209). The scale is based on the percentage of total leaf area infected and on the morphological development of the plant.

I. Induction of stagheads: Verma and Petrie (255) suggested two possible mechanisms of staghead development : 1) Early infection of young seedlings, starting with oospore-infested seeds and infection progressing systemically throughout the development of the plant, and 2) infection of young flower buds by zoospores arising from wind-borne sporangia. According to Verma and Petrie (255), oospores of *A. candida* race 7 mixed with seeds of turnip rape prior to sowing results in high levels of leaf and staghead infections. Over 55% staghead infections were induced by flower bud inoculation. Lakra and Saharan (126) although could not initiate staghead infection on *B. juncea* by inoculating flower buds with zoospores-sporangial suspension but were able to get >66% stagheads when 5 g/pot of oosporic inoculum was added with the seed at sowing time, along with subsequent sporangial spray inoculation at the seedling, branching and flowering stages of the crop.

Systemic infection was found to be directly correlated with the host age. With increases in age, a sharp decline in the ability to induce systemic infection occurs.

j. Germplasm screening: Many procedures have been described for evaluating host germplasm for resistance to white rust (61, 176, 252, 255).

1. **Detached leaf culture:** Verma and Petrie (253) transferred 12-14 day-old detached leaves of *B. campestris* to petri dishes containing 20-25 ml of autoclaved medium consisting of 0.5 ppm benzyladenine and 0.8% agar. Leaves were placed in the dishes with their lower surface on the medium usually within 15 min of detachment. Leaves were drop inoculated with a zoospore suspension (100,000 - 150,000/ml) derived from sporangia of *A. candida*. Leaves were incubated for 72 h at 100% R.H. with a day-night temperature regime of 21 and 16°C, respectively.

2. **Greenhouse screening:** At the time of sowing 5 g of oosporic material per pot is added to the soil. After emergence sporangial spray inoculations are made at the seedling, branching and flower stages. High humidity is maintained through frequent irrigations (126). Sporangial spray inoculation techniques have also been reported by several researchers (58, 61, 170, 176, 216, 227, 252, 254, 257).

3. **Field screening:** Verma and Petrie (255) were first to demonstrate the potential importance of seed infestation in initiating white rust. In their two years of field experiments, seeding oospores of *Albugo candida* race 7 with seeds of turnip rape (*B. campestris*) cv. Torch resulted in a significant increase over the control in both foliar and systemically-infected (stagheads) plants.

Recently, Saharan *et al.* (189) and Lakra and Saharan (126) established a white rust disease nursery by adding stagheads to the soil continuously for 3-5 years. When germplasm lines are sown, oosporic material is broadcast and also sown mixed with the seed. Frequent irrigations are given to maintain high humidity. Sporangial spray inoculations are made at the seedling, branching and flowering stages of the crop growth. Genotypes are usually grown in paired rows between highly susceptible "infector" rows of plants. Floral infections are produced by plucking the main shoot of the test row genotype

about 6" from the tip. This allows sporangial inoculum to fall on the test plants from the taller infector plants.

10. DISEASE CONTROL

a) Fungicides: Early work on the chemical control of white rust diseases focussed on the use of copper based fungicides to control the leaf phase of the disease. Frickhinger (63) and Naumann (153) reported that *Brassica* diseases can be controlled by frequent sprayings with copper fungicides. Vasudeva (248) recommended the use of Bordeaux Mixture or Perenox for the control of white rust of Brassicas and other diseases. With the progress in the development of dithiocarbamate, control of white rust was attained with multiple applications of protectant fungicides. However, these fungicides provided little protection from the staghead phase of the disease. Acylalanines, which are specifically active against Peronosporales, have now made it possible to control both the leaf and staghead phases of white rust either with a seed dressing or soil drenching and with fewer foliar applications. Three sprays of Polyram M @ 0.2% at 15 day intervals was most effective in controlling white rust of *B. campestris* var. Sarson in Pakistan (166).

Many fungicides have been evaluated for efficacy against white rust of *Brassica* crops in India. Benlate 0.1%, Calixin 0.1%, Difolatan 0.2%, Dithane Z-78 0.2%, Miltox 0.3%, Thiovit 0.3%, Mancozeb 0.2%, Mancozeb + Metalaxyl (Ridomil MZ 0.05%) and Ridomil 0.2% are effective in controlling both leaf and staghead phases of the disease and in increasing crop yield (3, 12, 66, 67, 73, 74, 111-114, 117, 123, 125, 186-188, 190, 191, 204, 210, 218). Three foliar applications of fungicide at 15 day intervals after the appearance of the disease, or at 40-45 days of crop growth, manages the disease effectively and economically (190, 191). For maximum disease control and high seed yield, treatment of seed with Metalaxyl (6 g Apron 35 SD/kg seed) followed by three sprays of Dithane M-45 or Metalaxyl was recommended (112, 187, 190, 191, 205). Seed treatment with Apron 35 SD protects the crop from white rust infection for at least 60 days (190, 191). To control the staghead phase, foliar sprays of Mancozeb, Metalaxyl, or a mixture of Mancozeb + Metalaxyl are required (114). Proper foliage cover and one spray coinciding

with flower initiation is very important to arrest secondary spread of the disease and flower bud infection (123, 125, 190, 191).

Seed treatment with mercurial fungicides may be effective to control white rust of rape (*B. campestris*) in Canada since these compounds inhibit oospore germination or kill the zoospores on emergence (254). Two applications of protectant fungicides like Bravo, Manzate 200, and DPX 164 reduced foliar infection in turnip rape (50, 254). Three sprays of acylalanine (Metalaxyl) fungicides, CGA 2912 and CGA 48988, were most effective in reducing staghead infections in rape (9, 50, 225, 226). Recently Stone *et al.* (227) advocated successful control of *A. candida* race 7 in *B. campestris* cv. Torch through foliar, seed and soil applications of metalaxyl. Foliar applications at 2.0 kg a.i./ha reduced systemic or staghead infection when applied at growth stages 3.2 or 4.1. To control staghead infection, a fungicidal spray at the flowering stage is essential. Seed dressing alone with metalaxyl at 5.0 g a.i./kg, or soil drench applications, reduced primary infections from germinating oospores. This subsequently reduced sporangial inoculum on the foliage during the growing season because metalaxyl uptake in rape is efficient through root absorption when applied to the soil. Using bioassays and chemical and gas chromatographic analyses of rapeseed plants grown from metalaxyl-treated seeds, and in metalaxyl-drenched soil, Stone *et al.* (228) showed that the fungicide was readily taken up by plants from the soil solution or from seeds. The greatest accumulation was in the lower leaves, with concentrations decreasing in the leaves furthest from the roots. Only small concentrations were in the stem and inflorescence. These results confirmed the observations of Sharom and Edgington (207), indicating that root absorption is a major factor in metalaxyl uptake.

Control of white rust of radish was most effective when four sprays of Difolatan 0.3%, Daconil 0.1%, Dithane M-45 0.2%, Ridomil 0.1% or Aliette 0.1% were applied at 8-10 day intervals (69, 91, 206). Dithane M-45 also increased yield significantly (205). The adsorption, mobility, and persistence of metalaxyl in soil and aqueous systems is greatly influenced by soil type, amount of rain and other environmental factors (207).

In the past Brestan, Captan, Bordeaux mixture and Zeneb were used to control white rust of horseradish (17, 63, 75, 101, 109). However, later studies showed that mancozeb and metalaxyl were more effective in controlling the disease and increasing the yield of horseradish (230).

Sprays of Captan and Cyprex have been used to control white rust of spinach (24). More recently, Jones and Dainello (97) suggest that effective control can be achieved with four sprays of Metalaxyl 0.28 kg a.i./ha, Chlorothalonil 1.17 kg a.i./ha, or EBDE (ethylene bisdithiocarbamate) 3.59 kg a.i./ha. Tank mixing reduced rates of Metalaxyl plus EBDC (0.15 + 0.58 kg a.i./ha) was as effective in controlling white rust as full rates of the individual compounds. Dainello and Jones (34, 35) studied the influence of continuous leaf wetness as a parameter for scheduling fungicide applications, and achieved the same level of control as the fixed schedule with Metalaxyl and Chlorothalonil.

In Brazil, copper fungicides are recommended to control white rust of *Ipomoea horsfolliae* (70). Deopel (49) suggested weekly sprays of Zeneb or Maneb @ 1 oz in 4 gallons of water to control white rust of Gerberas.

b) Cultural methods: To control white rust of cauliflower and horseradish, Savulescu (196) and Glaeser (69) suggested extirpation, the collection and burning of diseased plants in order to prevent the formation of oospores. Furthermore, these crops should be isolated from other crucifers. Reduction of relative humidity around the plants by adequate aeration, and avoiding of dense sowing and growth of weeds, also helps to reduce disease. The application of organic manures, avoidance of excess nitrogen and addition of phosphorus and potassium also increases resistance in plants.

Time of planting is very important for escaping staghead infection in *Brassica* crops. In India, early (September) and timely (mid October) sown crops escape staghead infection and suffer less disease in the leaf phase (114, 123, 131, 187). In Western Australia, crops of rape sown in early June generally have a higher incidence of stagheads, whereas crops sown in mid-July escape infection (6). This suggests that the incidence of stagheads in a crop is at least partially determined by environmental conditions.

c) **Resistance:** Control of white rust through host resistance has been attempted by identifying sources of resistance (42-45, 55, 56, 58, 170, 174, 176, 189, 239, 240, 275). All sources of resistance so far identified are race specific and governed by major genes. Zero erucic acid and zero glucosinolate *B. campestris* cv. Tobin, highly resistant to *Albugo candida* (race 7), was developed by the Agriculture Canada Research Station, Saskatoon, Saskatchewan, Canada and has been licensed for commercial use in Canada since 1980. Recently, Petrie (G.A. Petrie, personal communication), discovered occurrence of a race (Race 7a) to which cultivar Tobin was found susceptible.

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